# A Paper
ON
# ULCERATIVE COLITIS*

BY
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Sir Samuel Wilks, who in 1875 was the first to describe ulcerative colitis, pointed out that the condition was anatomically indistinguishable from dysentery. During the war I had frequent opportunities, at Lemnos and at Salonica, of sigmoidoscoping patients with dysentery. I recognized at once that amoebic dysentery presented a characteristic picture which bore no resemblance to that of bacillary dysentery, and that it was impossible to distinguish the latter from the ulcerative colitis with which I was already familiar.

## NATURE OF THE DISEASE

That infection with *B. dysenteriae* occurred in England was already well known from Eyre's investigations in 1894 on asylum dysentery. In 1912 Nabarro isolated the dysentery bacillus from a child who had been attending the Great Ormond Street Hospital for many years as a case of recurrent colitis. Since then he has found that not only many cases of summer diarrhoea, but many in small children diagnosed as chronic colitis, are caused by the dysentery bacillus. It is easy to understand how adults occasionally become infected with ulcerative colitis, due to *B. dysenteriae,* from children with these conditions, and yet do not themselves start a house epidemic, just as all the cases of amoebic dysentery arising in England which I have seen have been isolated ones. In 1923 Dudgeon isolated Flexner's bacillus in two cases of ulcerative colitis from material obtained from the surface of an ulcer examined through a sigmoidoscope. In 1927 Hadfield obtained it from the swab from an ulcer in a case I sigmoidoscoped forty-eight hours after the onset of haemorrhagic diarrhoea. The bacillus was isolated by Thorlakson and Cadman of Winnipeg in 1928, in six out of nine cases, from material obtained by scraping the base of an ulcer with a sharp curette, though they had never before discovered it in stools or in ordinary swabs from the ulcer surface. Last year Knott isolated Flexner's bacillus, which was agglutinated by the patient's serum, from a swab obtained from the raw surface left after I had snared a polypus in the rectum of a woman, aged 36, who had had very severe ulcerative colitis since 1925 and had developed two long strictures and multiple polyposis, although numerous examinations of stools and swabs taken from ulcers during the five years of her illness had only shown the usual organisms found in such cases.

It is well known that it is difficult to isolate the *B. dysenteriae* from the stools of patients in the Tropics suffering from typical dysentery after the acute stage has passed. The majority of cases of ulcerative colitis which come under skilled observation in England are already chronic, so that it can be easily understood how rarely the organism is found. In my only early case it was isolated without difficulty, and both Thorlakson's cases and the case I have just described show that it may be present in the depth of the lesion, when it cannot be demonstrated in stools or in swabs taken from the ulcers. These positive findings seem to me to be of more importance than many negative observations. The identical morbid anatomy and the response of many cases, especially early ones, to specific treatment with antidysenteric serum confirm the view which was first expressed by Saundby in 1906, and by Hawkins in 1909, and which I have consistently held since the war, that the disease is a form of bacillary dysentery.

Achlorhydria, which was generally secondary to gastritis, was present in about 25 per cent. of my cases. This is an important predisposing cause, as the absence of the normal acid antiseptic barrier of the stomach allows organisms swallowed in infected water or food to reach the intestines.

## COMPLICATIONS

I have realized only in recent years the frequency of certain serious complications of ulcerative colitis, which were never recognized before the war. This is probably due to the fact that numerous severely affected persons who would formerly have died now survive as a result of better treatment; many recover completely, but in others the disease runs a very chronic course, which gives time for these complications to develop.

Among 693 cases at the Mayo Clinic between 1923 and 1928 there were 69 patients with adenomatous polyposis, 59 with stricture, 26 with perirectal abscess, 18 with perforation, and 15 with malignant disease, in addition to 30 with arthritis (Bargen). This agrees closely with my own experience of the relative frequency of these complications, except that I have never seen a case of perforation. Though the ragged polypoid tags of mucous membrane mentioned by Dr. Cuthbert Dukes are often mistaken for true polypi, there is no doubt that true polypi also occur; some of those I have removed have been found on microscopical examination to be typical adenomata, and their *x*-ray appearance is identical with that of primary polyposis. In a few such cases the polypi have completely disappeared with deep *x*-ray treatment; but this should never be used until the primary ulceration has healed. It is most important to remember that strictures may develop without any change in the symptoms; in chronic cases a barium enema should therefore be given from time to time. I have had three cases in which complete recovery followed a short-circuiting operation, without which death would certainly have occurred. One patient has now been well for six years. There was stasis in the enormously dilated caecum and terminal ileum for seven days, although she was still having the usual severe haemorrhagic diarrhoea. In another case with an eleven years' history the strictures were associated with polyposis. I have seen two cases in which carcinoma apparently developed from polypi of the rectum following ulcerative colitis, and in another, carcinoma, polyposis, and active ulceration were all present together. Anal infections are of great importance, as they may persist after the colitis has healed, and may lead to reinfection of the colon.

## DIAGNOSIS

Direct examination of the mucous membrane of the rectum, which is almost always the part first affected and last to heal, is essential for diagnosis and for guidance in treatment. For those with limited experience a long proctoscope is easier to manipulate than a sigmoidoscope; it has the advantage of bringing the mucous membrane nearer to the eye, and the condition of the anal canal —a matter of considerable importance—can be more easily investigated. I have seen several cases of polypi and of carcinoma of the rectum and pelvic colon which had been treated as ulcerative colitis until I sigmoidoscoped them. I have recognized with the sigmoidoscope about half a dozen cases of amoebic dysentery in patients who had never been out of England and who, having hitherto failed to respond to treatment, recovered completely after twelve injections of emetine. I recently proctoscoped a patient who had been regarded as suffering from

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long-standing ulcerative colitis. The mucous membrane of the rectum was normal, the blood having come from haemorrhoids, and the lumen was filled with effervescing fluid faeces, which indicated that the diarrhoea was due to carbohydrate intestinal dyspepsia. Rapid recovery followed abstention from starch-containing vegetables.

### *X*-RAY INVESTIGATION

The *x* rays are very useful in investigating cases of ulcerative colitis. As the normal haustration of the bowel disappears in the affected part, it is possible, by means of a barium enema, to determine the extent of the disease. It is not uncommon for more or less of the proximal part to be spared in cases in which the pelvic colon and rectum are severely involved. When recovery takes place the haustration returns, except in very chronic cases. The proximal part of the colon is the last to be involved and the first to recover ; the disease almost always begins and ends in the rectum and pelvic colon, so that sigmoidoscopy detects the earliest stage of the disease, and when the mucous membrane of the rectum and pelvic colon are found to be no longer inflamed it may be assumed that the same is true of the whole colon. A barium enema also reveals the presence of strictures, but it is possible to distinguish between those caused by prolonged spasm and those caused by organic disease only by means of an opaque meal. Narrowing due to spasm does not cause any stasis, the colon being evacuated with great rapidity, whereas that due to a fibrous stricture leads to severe stasis in the dilated proximal segment. Lastly, the presence and extent of multiple polypi can often be recognized after an opaque enema or meal.

### TREATMENT

Rest in bed, warmth, and a generous mixed diet, from which the skins and pips of fruit and fibres of vegetables are alone excluded, are essential parts of treatment. When anaemia is present transfusion not only improves the general condition, but often greatly hastens the healing of the ulcers.

By absorbing gas, charcoal often makes the patient much more comfortable. Of the various drugs used locally I have found tannic acid (1 to 2 grains to 1 ounce) the most useful. If given after preliminary lavage with normal saline solution under low pressure through a soft catheter introduced just beyond the anus, it reaches the caecum, and is generally just as effective as if given through an appendix or caecal stoma. Quite recently I have been using dilute hydrogen peroxide (1 drachm to 1 pint) without preliminary lavage, and I am inclined to think this may prove even more satisfactory than tannic acid.

In the first case in which I used polyvalent anti-dysenteric serum in 1921, the result was little short of miraculous. A young man who was almost moribund after being very ill for over a year, and in whom no improvement had followed an appendicostomy, recovered completely in a fortnight ; five days after the first injection the sigmoidoscope showed that the innumerable ulcers seen a few days before had vanished, and nine days later the appearance of the mucous membrane was absolutely normal. Since then I have had a number of cases in which a dramatic recovery has taken place, and my colleagues Dr. Fawcett and Dr. Ryle, and Jerwood, Bindon Brew, and many others, have had similar experiences. Rapid recovery is most likely to occur in early cases, but it is occasionally observed in those which are very chronic. Thus, complete recovery occurred within a week in the very early case I have already mentioned in which Flexner's bacillus was isolated. More frequently the serum produces a certain amount of improvement, with the result that other treatment leads to recovery more rapidly than it would otherwise have done. In a small number of cases, especially very chronic ones, the serum appears to have no effect.

I have never seen the slightest benefit follow any form of vaccination, and in some cases the local condition has been definitely aggravated. Several American physicians have told me that they have been quite unable to confirm Bargen's enthusiastic reports about vaccination with his organism, which has not been accepted by any other American workers as the cause of ulcerative colitis.

In my experience the mortality of ulcerative colitis is extremely low. Complete recovery may occur after very long periods of treatment, and in cases which do not respond to serotherapy as much as a year or two of continuous treatment may be required. There is also an unfortunate tendency to relapse. The danger of this is much reduced if treatment is continued until the sigmoidoscope shows no trace of inflammation, even if symptoms have already disappeared for some weeks. Associated conditions, such as oral and pharyngeal infections and anal complications, must be treated, as a relapse may follow an acute sore throat or the development of a peri-anal abscess or a fistula in ano. The patient should always take sufficient paraffin to keep his stools soft ; if achlorhydria is present, the associated gastritis should be treated, and, if the secretion of acid does not return, the patient should take hydrochloric acid for the rest of his life.

### BIBLIOGRAPHY

Bargen, J. A.: *Ann. Int. Med.*, 1929, iii, 335.
Brew, R. Bindon: *British Medical Journal*, 1930, i, 478.
Dudgeon, L. S.: *Lancet*, 1923, i, 1007.
Eyre, J. W. H.: *British Medical Journal*, 1904, i, 1002.
Hawkins, H. P.: Ibid., 1909, i, 767.
Hurst, A. F.: *Guy's Hospital Reports*, 1921, lxxi, 26, and *Medical Essays and Addresses*, 1924, 150.
Jerwood, B. E.: *Guy's Hospital Gazette*, 1921, xxxv, 292.
Nabarro, D.: Personal communication, 1931.
Saundby, R.: *British Medical Journal*, 1906, i, 1325.
Thorlakson, P. H. T.: *Canadian Med. Assoc. Journ.*, 1928, xix, 656 ; and *Trans. Amer. Proctological Soc.*, 1930, xxx, 100.
Wills, S., and Moxon, W.: *Lectures on Pathological Anatomy*, second edition, 1875, p. 408.

# PULMONARY TUBERCULOSIS IN CHILDHOOD*

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*(With Special Plate)*

I think it is fair comment to say that in the past too little attention has been paid to the diagnosis and treatment of pulmonary tuberculosis in young children. The non-pulmonary forms of this disease have received more attention and they have given more satisfactory results. The provision of beds in special institutions for pulmonary tuberculosis occurring in children has not been, in relation to the number of cases, so generous as for the non-pulmonary types ; this, and the better response to treatment of the non-pulmonary forms, have been contributory factors to the present position. In making use of the term " childhood type " of tuberculosis, I refer to lesions in the lungs and intrathoracic glands which are the result of a first infection with the tubercle bacillus.

### INCIDENCE AND COURSE

Figures for the year 1923, published by the National Association for the Prevention of Tuberculosis, show that in England and Wales between 2,700 and 2,800 children

* Summary of an address read to the infant welfare subgroup of the Midland branch of the Society of Medical Officers of Health.